Computed tomography evaluation of aortic remodeling after endovascular treatment for complicated ulcer-like projection in patients with type B aortic intramural hematoma

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Objective: **The purpose was to investigate changes of the affected aorta after endovascular treatment for complicated ulcer-like projection (ULP), including aneurysmal change or rupture of ULP, or both, in patients with type B aortic intramural hematoma (IMH).**

Methods: **The study evaluated 18 patients (nine men) undergoing thoracic endovascular aortic repair for progressive aneurysmal formation of ULP (n = 17), rupture of ULP (n = 5), or both. Patients were a median age of 72 years (range, 45-83 years). Regular follow-up computed tomography studies were performed after treatment (mean follow-up, 61.2 months). A workstation was used to calculate changes on computed tomography images in the size or volume of ULP, affected aorta, and IMH. The growth rate of the volume of the affected aorta and IMH was also calculated before and after treatment. We evaluated the data using the paired *t*-test.**

Results: **A stent graft was successfully deployed and ULPs disappeared in all patients. IMH disappeared in 16 or decreased in two after treatment. There were significant differences in the mean maximum aortic diameter (37.8 ± 5.2 vs 34.5 ± 5.2 mm; *P* = .0006), mean IMH volume (39.4 ± 12.1 vs 2.0 ± 6.0 mL; *P* < .0001), and total volume of the aorta with IMH (158.1 ± 40.2 vs 128.9 ± 28.0 mL; *P* < .0001) before and after treatment.**

Conclusions: **Endovascular treatment is a useful treatment for complicated ULP in patients with IMH. Endovascular treatment also contributes to ideal remodeling of the affected aorta. (J Vasc Surg 2014;59:693-9.)**

Aortic intramural hematoma (IMH), one of the variants of aortic dissection (AD), has been considered as an important disease entity in the differential diagnosis of acute aortic syndrome.[1,2] Although diagnostic criteria of IMH have been established based on various imaging modalities,[3-5] their pathophysiologic link has not fully established.[6-10] In patients with IMH, ulcer-like projection (ULP), noted as a focal contrast enhancement in the thrombosed false lumen on computed tomography (CT), has been described.[4,11,12]

Recently, endovascular treatment in patients with ULP to prevent progression to double-barreled AD, aneurysm, or rupture, has been reported; however, no information is available about the outcomes of these patients after endovascular treatment. Endovascular treatment markedly improved IMH in several reports, but only a few patients were reported.[13] In addition, clinical implications of endovascular treatment for ULP have not been understood. The purpose of this study was to investigate changes of the affected aorta after endovascular treatment of complicated ULP, including aneurysmal change or rupture of ULP, in patients with IMH.

METHODS

This retrospective study was approved by the Nagasaki University School of Biomedical Sciences Institutional Review Board, which waived the informed consent requirement.

Study population. Between January 2004 and March 2012, 70 consecutive patients were admitted to our hospital with a diagnosis of acute type B IMH (not involving the ascending aorta). All 70 patients had chest pain or sudden back pain, and CT assessments were obtained ≤24 hours from symptom onset. IMH was defined on the CT image as crescent or circular hematoma in the aortic wall without direct flow communication from the true lumen, except for ULP. The CT studies were used to detect the presence of ULP, defined as a little contrast-enhanced island-like lesion within the IMH with direct communication from the true lumen or a localized blood-filled lesion protruding into the IMH. Precontrast and postcontrast CT images were used to differentiate ULP from penetrating atherosclerotic ulcer and to distinguish it from intimal calcification.[1]

The 70 patients were admitted, and intravenous antihypertensive drugs were used to control systolic blood pressure between 100 and 120 mm Hg. After stabilization,

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patients took oral antihypertensive agents to control systolic blood pressure at <120 mm Hg. Patients without complications, such as progression of IMH, ULP, double-barreled AD, and rupture, were discharged ≤1 month from the onset. The patients were monitored regularly after discharge. A regular follow-up CT study was performed ≤2 weeks and 2 months after discharge, followed by a CT study every 6 months to 1 year.

ULPs were seen during the follow-up period in 31 of 70 patients (44%) with type B IMH. Initially, medical treatment was performed for all 70 patients.

In two patients, ULP progressed to type A double-barreled AD. ULP in two patients progressed to aneurysm, but open surgery was selected because the ULP was identified in the proximal aortic arch. In one patient, ULP progressed to type B double-barreled AD, but medical treatment was selected because it was stable. The ULP in eight patients disappeared or was stable during the follow-up period. Finally, 18 patients (9 women and 9 men) with thoracic endovascular aortic repair were evaluated (Table I). They were aged between 45 and 83 years (median, 72 years; interquartile range, 53-70 years).

Our thoracic endovascular program was initiated in 1999 with the first intervention performed for AD; however, until 2008, when the first stent graft was commercially available, endovascular treatment was generally reserved for patients considered poor candidates for open repair. Since that time, endovascular treatment has been the option of choice for complicated ULP in patients with type B IMH if anatomically suitable, with open surgery reserved for nonendovascular treatment candidates. This study defined complicated ULP as (1) progressive aneurysmal dilatation (size of the aorta with ULP >1.5 times normal) or (2) rupture of the ULP, or both. We considered these findings as indications for intervention. Endovascular treatment was evaluated in a multidisciplinary fashion with the involvement of thoracic surgeons and interventional radiologists.

Endoluminal exclusion of ULP. All 18 patients underwent endovascular treatment through a femoral artery. Devices used included a homemade stent graft in six, TAG (W. L. Gore and Associates, Flagstaff, Ariz) in 10, and Zenith TX2 (Cook Medical, Bloomington, Ind) in two. The proximal site for the Zenith TX2 was in the healthy aortic wall. The homemade stent grafts were constructed of self-expanding stainless steel Gianturco Z-stents (Cook Medical) covered with ultrathin woven polyester (Dacron; DuPont, Wilmington, Del) graft material (thickness, 0.1 mm; porosity, 150 mL/cm^2/min/120 mm Hg; Ube Industries, Ube, Japan).

All procedures were performed in a unit with fluoroscopic and angiographic guidance. We did not use intravascular ultrasound or transesophageal echocardiography. General anesthesia with tracheal intubation was administered to all patients. An 8F sheath was inserted into the left femoral artery, and a 5F calibrated angiographic pigtail catheter was advanced into the ascending aorta to perform arteriography and permit arteriographic evaluation of the distance between the left subclavian artery and ULP. Arteriotomy was performed on the femoral artery originating from the true lumen. A 5F pigtail catheter and a 0.035-inch guidewire were advanced into the true lumen through this arteriotomy site until the ascending aorta was reached. A Lunderquist Extra Stiff Wire Guide (Cook Medical Inc) was advanced into the ascending aorta using the catheter exchange technique. After digital subtraction angiography was performed to confirm that the catheter was in the true lumen, the relative data of the aortic lesion were measured again, and the position of the proximal landing zone was marked on the screen. After intravenous administration of heparin sodium (0.5 mg/kg), the stent graft delivery system was advanced over the Lunderquist Extra Stiff Wire Guide under fluoroscopy and placed within the true lumen. When the proper position was reached, the systolic pressure was decreased to <80 to 90 mm Hg to ensure precise stent graft positioning. The stent graft was deployed by pulling back the sheath with the mandrel pusher firmly fixed.

The ULP was covered with the stent graft. The left subclavian artery or left common carotid artery, or both, were covered for adequate coverage of the ULP. We did not cover all sites of IMH. Each patient was treated with one endograft prosthesis. The left subclavian artery was intentionally occluded in two patients after an occlusion test of this artery showed collateral circulation to the left upper limb from the left vertebral artery. Both left subclavian and left common carotid arteries were intentionally occluded in one patient after right subclavian-to-left common carotid artery bypass was performed.

Table I. Patient characteristics, clinical profile, computed tomography (*CT*) examination, and outcome in 18 patients

Parameter	*No. (%) or median (range)*
Male-to-female ratio	9:9
Age, years	
Total	72 (45-86)
Men	66 (70-86)
Women	79 (43-82)
Hypertension	16 (90)
Diabetes	5 (28)
Renal failure	3 (17)
Current smokers	8 (50)
CT scans per patient	5 (3-10)
CT follow-up after endovascular treatment, months	61.2 (0.3-131)
Stent graft	
Homemade	6 (33)
Gore TAG[a]	10 (56)
Zenith Tx2[b]	2 (11)
Complication due to endovascular treatment	2 (11)
Deaths[c]	3 (17)

[a]W.L. Gore and Associates, Flagstff, Ariz.
[b]Cook, Bloomington, Ind.
[c]All deaths occurred after endovascular treatment.

Angiography was performed immediately after deployment to confirm coverage of ULP and blood flow of the aortic lumen and branch vessels. After removal of the large sheath, the arteriotomy was repaired.

Analysis of CT and clinical data. CT was performed using Somatom Sensation 16 (Siemens Medical Solutions, Forchheim, Germany) or Somatom Definition (Siemens Medical Solutions). All axial CT images were obtained in a contiguous 1- to 5-mm-thick section. Unenhanced and enhanced images were obtained from the thoracic inlet to the inguinal level in the craniocaudal direction. Coronal, oblique coronal, and sagittal reformatted images were also obtained. An automated injector was used to administer 100-mL iomeprol contrast material (Iomeron300; Bracco, Milan, Italy) intravenously at a rate of 3 mL/s, followed by a saline chaser.

Three-dimensional reconstruction software, Aquarius iNtuition 4.4 (TeraRecon, San Mateo, Calif), was used to measure the IMH and aorta with IMH volumetrically. On three-dimensional images reconstructed from CT Digital Imaging and Communications in Medicine (DICOM) data, the free region-of-interest tool was applied to define the aorta and IMH. With slice-by-slice definition, the software generated volume information about the selected vessel semiautomatically.

The aortic arch was defined as the segment between the brachiocephalic artery and the estimated ligamentum arteriosus. The descending aorta was defined as the segment between the estimated ligamentum arteriosus and the aortic hiatus of the diaphragm.

CT images were evaluated by two experienced cardiovascular radiologists (E.S., I.S.), each with >10 years of experience. Final decisions regarding the findings were reached by consensus.

Data retrospectively obtained by reviewing the medical records included patient symptoms and signs, CT results, therapeutic modality, course of treatment in the hospital, and follow-up outcomes until the end of 2012. After endovascular treatment, the mean CT follow-up period was 61.2 ± 45.5 months (range, 0.3-131 months).

The CT measurements were used to calculate changes in aortic size on multiplanar reconstruction images using a workstation. The orientation of the multiplanar reconstruction image was manipulated to obtain a plane parallel to the aorta, and the largest short axial diameter of the outer contour of the affected segment of aorta was measured. Double-oblique cross-sectional sections perpendicular to the long axis of the aortic lumen were used to measure wall thickness using the workstation calipers, expressed in millimeters. The maximal thickness of the IMH was measured in the aortic arch, descending aorta, and abdominal aorta after selecting the site with maximal wall thickness by visual inspection.[1] We measured the transmural, circumferential, and longitudinal diameters of each ULP and developed ULP. The transmural diameter of the ULP was the maximal thickness of the ULP perpendicular to the aortic wall, the circumferential diameter was the maximal width of the ULP length parallel to the curvature of the aortic wall, and the longitudinal diameter was the maximal longitudinal cephalocaudal length of the ULP.[14] In all patients, the final CT before endovascular treatment was obtained ≤48 hours before the procedure.

The growth rate of the volume of the affected aorta and IMH was calculated in the following manner: The difference in the volume between ($V1$) and ($V2$) measurements was divided by the time interval (T) between the two measurements:

Growth rate of volume = $(V2 - V1)/T$.

Finally, the growth rate per week before and after endovascular treatment was calculated in the following manner: The difference in the volume between the initial ($V1'$) and final ($V2'$) measurement before endovascular treatment and the difference in the volume between the final ($V2'$) measurement before endovascular treatment and final ($V2$") measurement after endovascular treatment. The time of final ($V2$") measurement after endovascular treatment was when the IMH disappeared.

Growth rate of volume before endovascular treatment = $(V2' - V1')$/1 week (7 days).

Growth rate of volume after endovascular treatment = $(V2" - V'2')$/1 week (7 days).

Statistical analysis. Continuous variables are expressed as the mean ± standard deviation. Statistical analysis was performed using clinical and morphologic variables with the paired t-test and Mann-Whitney U test for continuous variables. In all tests, $P < .05$ was considered significant. SPSS 11.5 software (SPSS, Chicago, Ill) was used for analysis.

RESULTS

In 18 patients, IMH was involved as follows: from the aortic arch to abdominal aorta in 3, from the aortic arch to descending aorta in 11, and descending aorta in 4. In six of 18 patients, ULPs were identified at initial CT. In 12 patients, ULPs appeared during the follow-up period from 1 to 10 days after onset. Three of 18 ULPs were located in the distal aortic arch, and 15 were in the descending aorta.

Demographics and clinical factors of patients are reported in Table I. Patients underwent endovascular treatment from 7 to 91 days after onset. In 18 endovascular treatments, 17 (94%) were performed for progressive enlargement of ULP or in 5 (28%) for rupture, or both.

The stent graft was successfully deployed in all patients. All ULPs were closed by the stent graft. No complications occurred during procedures.

During the follow-up period, no patients had any recurrence of ULPs after endovascular treatment. The size of ULP was 20.1 ± 6.4 mm before endovascular treatment and 0.0 ± 0.0 mm after treatment (Fig 1), with a significant difference ($P < .0001$).

In two of 18 patients (11%), additional aortic injury occurred during the follow-up period, which was diagnosed by CT. One patient had retrograde AD 2 years after endovascular treatment. This patient had no symptoms and refused surgery. One patient had pseudoaneurysm formation 6 months after endovascular treatment (Fig 2).

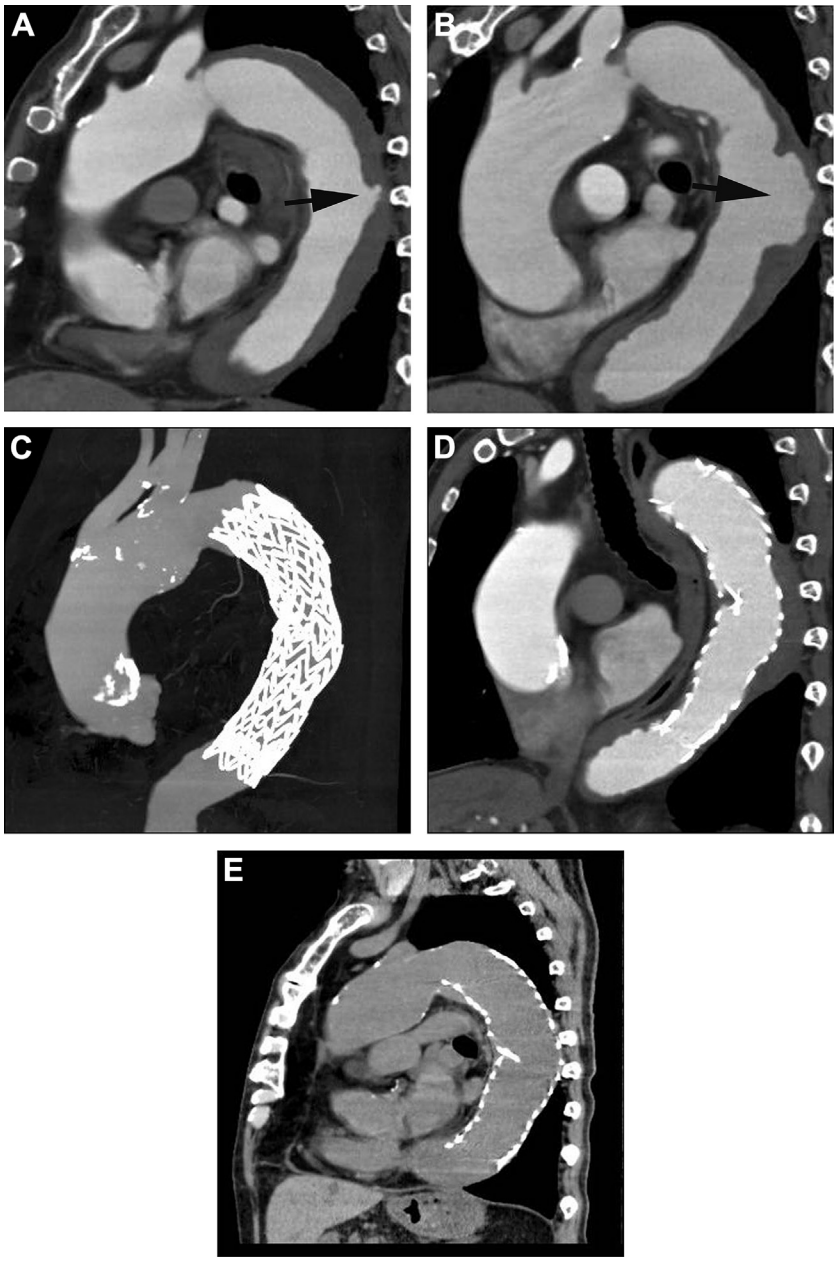

Fig 1. An 81-year-old woman with type B acute intramural hematoma (IMH) of the aorta. **A,** At onset, contrast-enhanced computed tomography (CT) images show the ulcer-like projection (ULP) in the descending aorta (*arrow*). **B,** A contrast-enhanced CT image 2 months after onset shows an enlarged ULP in the descending aorta (*arrow*). **C** and **D,** Contrast-enhanced CT images 2 weeks after endovascular treatment show that the ULP has disappeared. IHM has also decreased in size. **E,** A nonenhanced CT image 1 year after endovascular treatment shows that the IHM has disappeared.

The pseudoaneurysm was repaired in an additional endovascular treatment. No other complications due to endovascular treatment occurred during the follow-up period. No patients had a perioperative stroke or permanent paraplegia. No endoleaks or ULP occurred.

Three patients (17%) died during the follow-up period, one patient each of pneumonia, abdominal aortic rupture, and cerebral infarction at 10, 45, and 823 days from onset, respectively. Although the causes of death were not related to the endovascular treatment, one patient was classified as a perioperative death.

Changes of the aorta with IMH before and after endovascular treatment

Before endovascular treatment. In all patients, IMH was persistent before endovascular treatment. A comparison

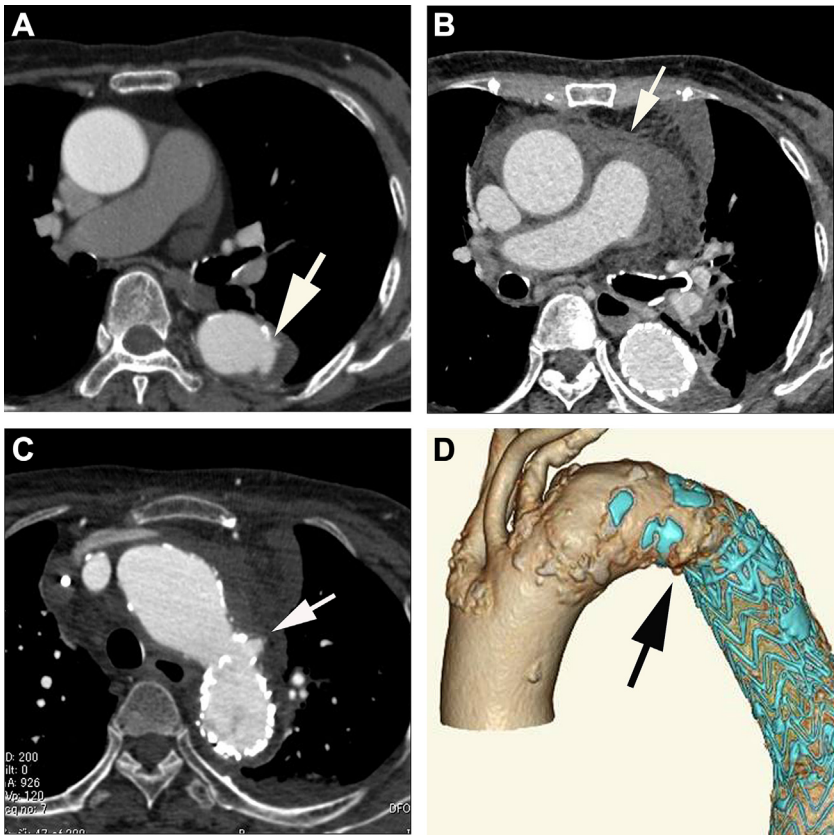

Fig 2. An 83-year-old woman with type B acute intramural hematoma (IMH) of the aorta. **A,** A contrast-enhanced computed tomography (CT) image 3 months after onset shows progressive ulcer-like projection (ULP) in the descending aorta (*arrow*). **B,** She had severe chest pain 1 year after endovascular treatment. A contrast-enhanced CT image shows that ULP had disappeared; however, mediastinal hematoma is seen (*arrow*). **C,** Contrast-enhanced CT images show a pseudoaneurysm caused by the edge of the stent graft in the aortic arch (*arrow*). **D,** CT angiography shows pseudoaneurysm caused by the edge of the stent graft in the aortic arch (*arrow*).

of the initial and final CTs before endovascular treatment showed the maximum thickness of false lumen IMH in four patients (22%) increased between initial and final CT before endovascular treatment. The mean maximum thickness of the false lumen at the initial and final CT before endovascular treatment was 8.9 ± 4.2 and 10.8 ± 4.3 mm, respectively, with no significant difference ($P = .0957$). There were no significant differences in the mean maximum aortic diameter (37.9 ± 6.8 vs 37.8 ± 5.2 mm; $P = .944$), mean IMH volume (42.5 ± 13.1 vs 39.4 ± 12.1 mL; $P = .844$), and total volume of the aorta with IMH (164.2 ± 30.2 vs 158.1 ± 40.2 mL; $P = .0755$) at the initial and final CT before endovascular treatment (Fig 1; Table II).

After endovascular treatment. A comparison of final CTs before and after endovascular treatment showed the IMH disappeared from 60 to 90 days after endovascular treatment in 16 patients (89%). In the remaining two patients (11%), the maximum thickness and volume of IMH decreased after endovascular treatment. The mean maximum thickness of the false lumen at final CTs before and after endovascular treatment was 10.8 ± 4.8 and 0.9 ± 2.4 mm, respectively, with a significant difference ($P < .0001$). There were also significant differences in the mean maximum aortic diameter (37.8 ± 5.2 vs 34.5 ± 5.2 mm; $P = .0006$), mean IMH volume (39.4 ± 12.1 vs 2.0 ± 6.0 mL; $P < .0001$), and total volume of the aorta with IMH (158.1 ± 40.2 vs 128.9 ± 28.0 mL; $P < .0001$) at final CTs before and after endovascular treatment (Fig 1; Table II).

Growth rate of volume of false lumen and total aorta before and after endovascular treatment

The growth rates of the mean false lumen volume before and after endovascular treatment were −3.3 ± 4.1 and −7.4 ± 5.7 mL/wk, respectively, with a significant difference between ($P = .0178$). There were no significant differences in the growth rates of total volume of the aorta with IMH (−4.0 ± 4.8 vs −5.4 ± 4.6 mL/wk; $P = .5513$) before and after endovascular treatment.

DISCUSSION

Current indications for endovascular treatment of type B IMH generally cover patients who have disease

Table II. Aortic remodeling parameters of computed tomography (*CT*) measurement before and after endovascular treatment (*ET*)

Variable	*Initial CT before ET, mean ± SD*	P[a]	*Final CT after ET, mean ± SD*	P[b]	*Final CT, mean ± SD*
IMH					
Thickness, mm	8.9 ± 4.2	.0957	10.8 ± 4.3	<.0001	0.9 ± 2.4
Volume, mL	42.5 ± 13.1	.844	39.4 ± 12.1	<.0001	2.0 ± 6.0
Max aortic diameter with IMH, mm	37.9 ± 6.8	.944	37.8 ± 5.2	.0006	34.5 ± 5.2
Total volume of affected aorta, mL	164.2 ± 30.2	.0755	158.1 ± 40.2	<.0001	128.9 ± 28.0

IMH, Intramural hematoma; *SD*, standard deviation.
[a]For comparison of data for initial CT data vs data for final CT before ET.
[b]For comparison of data for final CT before ET vs data for final CT after ET.

progression or complications such as repeat dissection, (contained) rupture, persistent chest pain, or patients unresponsive to antihypertensive treatment. Several studies showed that endovascular treatment was performed for penetrating atherosclerotic ulcer in patients with IHM, but there are few reports in which endovascular treatment was performed for complicated ULP in patients with IHM.

ULP is defined as a localized contrast medium-filled pouch in the hematoma of the false lumen, which obviously communicates with the true lumen.[14-18] ULP has been considered as a site of intimal disruption. These lesions can develop to localized AD and aneurysmal dilatation.[1,14-19]

In our study, all complicated ULPs were successfully closed and disappeared after endovascular treatment, which suggests that endovascular treatment can be a useful treatment for complicated ULP. The proven safety and efficacy of endovascular treatment have led to its expanding application to a wide variety of thoracic aortic pathologies[20]; however, for complicated ULP, a generally accepted therapeutic strategy has not been established, yet. Further studies are therefore needed.

According to previous reports, IMH showed various remodeling processes from complete resolution to complications such as development of aortic rupture, repeat dissection, or aneurysmal dilatation.[1,8,9,21,22] IMH may be a more dynamic condition or more vulnerable than double-barreled AD.[8] Generally, initial medical treatment, without surgical intervention, is selected for stable patients with type B IMH.[9,21] However, because of the variable remodeling processes in patients with type B IMH, investigations of long-term outcomes to determine prognostic factors are needed.

In this study, IMH disappeared or decreased in size after endovascular treatment in all patients. In addition, there were significant differences in the mean maximum aortic diameter (37.8 ± 5.2 vs 34.5 ± 5.2 mm; $P = .0006$) before and after endovascular treatment.

Previous reports have shown IMH can progress with a significantly lower long-term survival rate. The incidence of IMH progression was 28.0%.[9,23] A previous report revealed a significant association between ULP and development of IMH,[1] which suggests that ULP may provide blood flow or arterial pressure to the false lumen.

Our results also revealed that closed ULP by endovascular treatment was related to hematoma resorption of the affected aorta. This supports that ULP may provide blood flow or arterial pressure to the false lumen. If endovascular treatment closes the ULP, blood flow or arterial pressure of the false lumen decreases or disappears. This mechanism may contribute to ideal remodeling of the affected aorta. A previous report showed that the appearance of an ULP is predictive of progression (development of double-barreled AD, increased hematoma thickness, or aortic enlargement) in patients with type B IMH. This report may support our speculation.[10] In patients with penetrating atherosclerotic ulcers, we speculate that endovascular treatment may contribute to ideal remodeling of the affected aorta by the same mechanism.

According to previous studies, aneurysmal dilatation is seen in 53% of patients with IMH during the chronic phase. This complication has also been reported previously,[9,23] along with a progressive aneurysm development, even with complete resolution.[24] Because the aortic wall is damaged by IMH, aneurysmal change can occur in the affected aorta during the chronic phase.[7] This study did not show long-term results of the affected aorta treated by endovascular repair. Further long-term studies are needed to clarify the results of the affected aorta treated by endovascular treatment.

Two of 18 patients presented with additional aortic injury, but stent graft causation of these lesions was not confirmed. Recent reports showed that a stent graft-induced new entry (SINE) tear developed preoperatively or during follow-up in patients with double-barreled AD. SINE was defined as a new intimal tear damaged by the stent graft itself, excluding those created by natural disease progression or any iatrogenic injury.[25,26] Proximal and distal SINE represented SINE at the distal and proximal ends of the endograft, respectively, with an incidence of 3.2% and a mortality rate of 26.1%.[25] Our study showed that a stent graft-induced aortic injury may occur in patients with IMH as a late complication. We have to be aware of these complications after endovascular treatment.

This study had some limitations. First, the number of patients was small, and the follow-up periods varied. Further long-term studies involving larger numbers of patients are needed.

Second, we used various types of stent grafts, which may have a potential bias; however, no episodes based on the type of stent graft were documented in this study.

CONCLUSIONS

This study revealed that endovascular treatment is a useful treatment for complicated ULP in patients with IMH. Endovascular treatment also contributes to ideal remodeling of the affected aorta, because closure of ULP may interrupt blood flow or arterial pressure to the false lumen; however, literature on the long-term efficacy and success of endovascular repair of IMH are still lacking. Further long-term studies involving a larger sample are needed.

AUTHOR CONTRIBUTIONS

Conception and design: ES, NH, KH, IS KE, MU
Analysis and interpretation: ES, NH, KH, IS
Data collection: ES, NH, KH, IS
Writing the article: ES, NH, KE, MU
Critical revision of the article: ES, NH, KH, IS, KE, MU
Final approval of the article: ES, NH, KH, IS, KE, MU
Statistical analysis: ES, NH, KH, IS
Obtained funding: ES
Overall responsibility: ES

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